

therapy that does not threaten the patient with a cure often appears to be the unexpressed attraction to the program. Names, prefixes, suffixes and adjectives create disease entities which then take on a life of their own, conjugate among themselves and end up with strange hybrids in which the ancestral features are obscured. Fibrositis (and its many nominal forebears and descendants) represents an example of how the common aches, pains, sorenesses—and sorrows—of humankind are, through naming, established as disease entities. Is this medical progress?

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Dr. Bennett Replies

TO THE EDITOR: Like Dr. Weinberger, there are many physicians who regard fibrositis as a "psychophysiologic state." That term itself is merely another example of "verbal juggling" and may ease the diagnostic conscience, but it does little to ease the patient's suffering. It is easy to dismiss a patient whose pain cannot be readily explained as a *crock*; it certainly saves the physician time and nervous energy, for the patient goes elsewhere. Pain is, of course, a purely subjective sensation; we have no way of measuring it effectively. There is no evidence that patients who have pain of a psychogenic origin suffer any less distress than those who have a well-defined cause for their pain—to think otherwise is pure arrogance.

One major advance in studying patients with fibrositis has come from the work of Smyth and Moldofsky.¹ They have set forth criteria that help to delineate patients with fibrositis from those with other similar pain syndromes; these criteria were amplified in my article. This, at least, is a start in the scientific study of this group of patients, for it enables investigators to compare results in similar patient populations. Such criteria

should not be misconstrued as implying that fibrositis is a distinct disease entity; that distinction can only be made if reproducible pathology (psychological or organic) is discovered.

Weinberger's contention that no pathology has ever been found and that, therefore, the term fibrositis should be abandoned is hardly in accord with the spirit of scientific curiosity that leads to new insights. One need only look at the history of medicine to see the folly of such thinking. To wit, in polymyalgia rheumatica (PMR), a disease with many symptoms in common with fibrositis, there is only one major abnormal laboratory test finding: an elevated sedimentation rate. If we were not cognizant of this fact, should we also call PMR a "psychophysiologic state"? Indeed, the ubiquitous history of early morning stiffness and the exacerbation of the symptoms of fibrositis by changes in the atmospheric pressure are so reminiscent of many of the better defined rheumatic disorders that one has to be skeptical of any purely psychophysiologic explanation.

I suspect that if modern medical training paid more attention to "the common aches, pains, sorenesses . . . of humankind," rather than emphasizing the unusual and the esoteric, the burgeoning paramedical pseudosciences would have fewer devotees. Fibrositis, whatever it is, is just such a problem. Finally, I would remind Dr. Weinberger of some words of Claude Bernard: "It is what we think we know already that often prevents us from learning."

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Acetylcholine Replacement Therapy in the Management of Huntington's Disease

TO THE EDITOR: Evidence suggests that the choreiform manifestations of Huntington's disease (HD) are caused by deficient acetylcholine synthesis in striatal neurons.¹ This hypothesis was tested in the management of two cases of HD that we have followed for longer than a year. Transmitter replacement of central cholinergic synapses was achieved orally by the combined administration of an acetylcholine precursor (lecithin) and a cholinesterase inhibitor, pyridostigmine bromide (Mestinon). Pyridostigmine bromide was chosen,

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rather than physostigmine, because it is less toxic than physostigmine and it is available in a convenient and palatable form.

The first patient was a 37-year-old white woman with established HD of seven years' duration (notable choreiform manifestations with persistent involuntary rotatory movements of the neck, irregular fidgeting of the hands, prancing gait with pronounced swaying of torso when walking). This patient received 60 mg per day of pyridostigmine bromide given orally in concentrate form (Mestinon) and 12 grams per day of lecithin in capsules.

After four weeks of therapy a gradual reduction in range and amplitude of the choreiform movements became apparent. The patient was able to groom herself and walk unassisted. This improvement persisted as long as the pyridostigmine-lecithin therapy was continued. The deliberate interruption of this regimen, four months after initiation of therapy, induced the reemergence of the choreiform symptomatology in its unmodified form. Upon resumption of therapy, chorea gradually subsided and was brought under control within three weeks. This patient has been maintained on this regimen for 30 months without any ill effects or overt signs of toxicity or degeneration of other systems. She has been able to gain considerable weight (8 pounds), partly owing to the ability to feed herself more adequately.

A second patient, with strikingly similar clinical features, responded equally well to this therapeutic regimen. Within three weeks choreiform movements were reduced by 80 percent and the patient was able to feed himself and gained 14 pounds.

We have reason to believe, therefore, that acetylcholine replacement therapy can be achieved successfully by the oral administration of an appropriate acetylcholine precursor and a cholinesterase inhibitor. These pharmacological manipulations appear to be safe and quite effective in the management of choreiform components of HD. The response is rather slow in onset and is seldom evident before four to six weeks from the onset of therapy. These results are permanent and stable provided that this regimen is maintained.

In summary, it appears that transmitter replacement of central cholinergic synapses can be a practical routine and that it can be achieved with relative impunity by the simultaneous administration of an acetylcholine precursor and a cholinesterase inhibitor. In 1978 Barbeau² advocated the use of choline or lecithin as a possible means

to replenish central cholinergic synapses. There are several neurological conditions for which there is presumptive evidence that some of the manifestations of the disease are the expression of a central cholinergic deficit. Huntington's chorea, Friedreich's ataxia, presenile dementia and Gilles de la Tourette's syndrome seem to fall into this category.

I believe that lecithin replacement, per se, is only weakly effective though assuredly safe. Its actions, however, can be enhanced by a moderate degree of inhibition of brain cholinesterase that can be achieved by the oral route with pyridostigmine bromide in doses ranging from 60 to 120 mg per day.

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Stewards of the Environment

TO THE EDITOR: The comments in the July editorial "There Will Be Changes Down the Road"¹ were intriguing. Although I generally agree that our government has spent money with abandon, the issues we face in health care, in our support of the elderly and in the continuation of medical research must be faced and appropriate alternative solutions sought.

I must, however, disagree with the statement that there is Biblical support for how mankind has reacted in his environment. Nowhere does there appear in that document commands or even suggestions to licentiously procreate or *exploit* our earth. To the contrary, if carefully studied, the Bible explicitly states that we humans are to be stewards of our environment; it indicates that God put us here to use the earth, not to abuse it. I am firmly convinced that the end of our world will occur when man has used up the earth and we perish in our own stupidity. I would hope that all physicians would in fact carefully use the Biblical guidelines which "make as much sense now as

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